MUSCULAR FATIGUE, MUSCLE STRAIN AND MUSCLE CRAMPS*

By RUDOLPH MARX, M. D.
Los Angeles

THE muscle as an energy-producing engine equals in efficiency the best man-made motor. Up to 35 per cent of the chemical energy used up during its action is transformed into useful power—the same as in the Diesel motor. The comparative efficiency of a modern steam-engine is only about 25 per cent; of a gasoline motor it is still less.

The main sources of muscular energy are glycogen and phosphagen. During exercise glycogen is changed into lactic acid, and phosphagen is broken down into creatinin and phosphoric acid. This process is accompanied by an increase of acidity. However, during the period of "recovery" these chemical processes are reversed, as in the charging of a battery. Under normal conditions this restitution is accelerated and completed by addition of oxygen.

MUSCULAR FATIGUE

Accumulation and insufficient removal of the decomposition products of muscular activity lead at first to a transient condition of increased irritability, followed by lessened irritability of the muscle cells. The latter state of more or less complete loss of muscular irritability and contractability is called "fatigue."

After the appearance of marked fatigue, a muscle usually shows improvement of irritability if it is given a short rest. But depending upon the age of the individual and condition of "training," intervals of some hours and even days may be required before a fatigued muscle regains the full capacity to perform its work.

If the fatigue-creating substances of a muscle overflow into the general circulation, or are experimentally injected into animals, they produce symptoms of general fatigue. The first effect is the depression of other muscles, including the muscles of the heart and blood vessels. This in turn leads to a diminution of circulation and oxidation and the vicious cycle of added fatigue. The functioning of the nerve cells and the central nervous system also suffer—directly by the fatigue substances and indirectly by diminished oxidation (except the breathing center, which is stimulated by the increased carbon-dioxid tension). This results in the impairment of muscular power, tempo and coördination of intentional and unintentional motion and motion sequences.

By repeated exercise ("training") the muscle is made to gain in size, tonus, strength, and power of recuperation. Its fibers grow larger, storing up more glycogen; the size of its blood vessels and the amount of blood circulating in it increase, and the acid-binding power of the muscle proteins seems to grow. (The muscles of the heart respond in a similar way.)

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MUSCULAR STRAIN

If stimulation of the muscle is continued after the state of fatigue is reached the muscle is apt to be damaged and "muscular strain" results. After the muscle has used up its own reserve material, eventually other glycogen brought up from the liver, and also its alkaline buffer substances, less suitable protein material of its cells may be called upon to produce energy and chemical balance. The evidence of this fact is the increased nitrogen output which results from exhausting exercise. If these destructive processes are permitted to go beyond a certain point, they produce more or less far-reaching physicochemical alterations in the muscle tissue. These changes are often accompanied by an inflammatory reaction, edema, and stiffness. The popular name for this condition of strain of muscles and muscle groups is "charleyhorse." The extreme possible consequences of such a state are muscular atrophy and contraction, which, however, occur only in rare instances.

In this connection it may be interesting to mention that the physicochemical alterations in the muscle substance, produced by extreme strain, seem to approach closely the changes seen in postmortem rigor. It is known that the muscles of persons dying after exhaustive efforts or from tetanus develop the condition of rigor mortis immediately or shortly after death; whereas the same process normally takes several hours to develop.

Muscular strain is apt to leave the muscles in a condition of increased irritability, with a tendency to cramps. By the term "muscular cramps" we understand involuntary painful spasms of muscles.

MUSCLE CRAMPS

Muscle cramps are usually brought on by sudden, exaggerated or wrongly directed impulses when a muscle action does not meet the anticipated amount of resistance or is not checked by the controlling antagonistic muscles, as is normally the case. After producing a maximum contraction the superfluous amount of muscle energy liberated by the disproportionate impulse is converted into a muscular spasm. This implies that a muscular spasm surpasses in intensity a voluntary muscular contraction. That part of the energy produced by it which is not converted into labor is transformed into heat.

The disposition to involuntary sudden contractions is increased in muscle groups which are in an advanced state of special training. They are conditioned to immediate response; their action has become almost automatic and reflex-like, and the slightest stimulus can make them contract. Then, as mentioned before, the irritability of muscles is also increased by a certain concentration of fatigue products.

In swimming the inhibiting effect of the cold on the peripheral circulation can also contribute to the disposition to cramps. The diminished circulation may be insufficient for the oxidation and removal of waste material in muscles, leading to ischemic pain and tendency to spasms.

PREVENTION AND TREATMENT

It is easy to understand that prevention of the conditions mentioned is only possible to a limited degree. And little can be said about their prevention and treatment that experienced trainers do not know. The theories merely support the practical rules of training which were found empirically.

To mention these briefly. The training work should be started with small demands and increased gradually. Sufficient rest periods must be interspersed. No effort in training should be carried to the point of complete muscular exhaustion. Once the muscles have reached a state of severe fatigue or strain, they should not be exercised until they have fully recuperated.

The process of recovery can be accelerated by the accepted methods known to stimulate the recuperation and circulation of the muscles, *i. e.*, heat in any form, and massage.

1930 Wilshire Boulevard.

THE OVARY OF THE RAT AFTER HYPOPHYSECTOMY

By Olive Swezy, M. D.
and
Richard I. Pencharz, M. D.
Berkeley

Much former experimental work has shown that the functioning of the ovary depends upon the presence of the hypophysis and its hormones. In another paper of this series it will be shown that an excess of these hormones has a depressing effect upon the growth of follicles and the production of new germ cells. A further study of the hypophysectomized rat reveals the rather surprising fact that the hypophyseal hormones in normal amounts, so necessary for follicular growth, also depresses ovogenesis or the production of new germ cells.

Eight rats were hypophysectomized at varying times from twelve to ninety days before autopsy (see Table 1). The ovaries of these were serially sectioned and stained with uniform procedure for each. The ova and primordial follicles in the entire ovary were counted, with the few larger follicles and corpora that were present, a single ovary being used from each rat. The results of these counts are shown in the appended table.

After hypophysectomy the ovary of the rat rapidly shrinks to a small size. In the ovaries we have studied, from four to thirty corpora lutea were present, the remainder of the ovary being filled with interstitial tissue and primordial follicles and ova. The corpora are remarkably persistent structures in these ovaries. Smith[1] found that corpora one-half millimeter in diameter were still in evidence at nine and one-half months after the operation. Although, in our own rats, evidences of regressive changes have appeared in all of the corpora, yet the size undergoes relatively little diminution in the first fifty days following the operation. In rat 1130, the largest corpus was about one-half that of the early part of pregnancy.

Table 1.—*Ova and follicles in hypophysectomized rats.*

Rat	Days After Hypophysectomy	Ova and Primordial Follicles	Corpora	Total	Age
5976	12	2822	30	2852	117
5929	20	4311	23	4334	95
4343	35	4100	30	4130	123
1121	50	3927	4	3931	166
8643	50 right	4681	22	4703	
	64 left	4969	41	5010	
1141	62	4715	9	4724	186
1081	79	3605	17	3622	203
1130	90	4345	8	4351	202

Those that were pregnant at the time of the operation show slightly larger corpora than were present in the nonpregnant rats. The number decreases by the end of fifty days, those that persist being probably the last "crop" which ovulated.

Interstitial tissue increases with the gradual disappearance of the older corpora and large follicles in these ovaries, and, in most cases, shows degenerative changes in the nuclei. In addition to that formed from degenerating follicles and corpora, the germinal epithelium also adds to the supply by the formation of groups of cells which are indistinguishable from other interstitial cells after they have severed all connection with the epithelium. This is relatively rare and is the same process that occurs normally during both the pregnant and nonpregnant periods.

The production of new germ cells is abundant in the ovary of the hypophysectomized rat. When the number of these was counted, it was found that the amount of ovogenesis, as shown by the number of ova and primordial follicles, was greater than is found in the ovary during the normal oestrous cycle or in pregnancy, the number produced being from two to three times that found in the normal rat. These counts are shown in the table. The germinal epithelium shows active proliferation of single ova as well as epithelial cords from which ova are developed, processes similar in every respect to those found in the normal rat.[2]

In the small follicles, which may occasionally reach a fairly large size (270 microns in diameter in rat 1130 at day 90), with six or eight or even more rows of cells in the granulosa, growth, as shown by the number of mitoses in the cells of the granulosa, seems to be about as rapid as in the normal ovary. As many as a dozen or more mitotic figures may be seen in one section of a follicle 200 microns in diameter. These evidences of normal growth in small follicles, combined with the amount of active proliferation from the germinal epithelium, seem to establish the fact beyond doubt that the large number of ova and primordial follicles found in these ovaries is not an accumulation over long periods of time, but is the result of an actually greater rate of ovogenesis than occurs in the normal rat. This increase evidently begins a short time after hypophysectomy and seems to be due to the withdrawal of the hormones of the hypophysis.